What is claimed is:

1. An isolated and purified nucleic acid sequence having the sequence set forth in SEQ ID NO:2.

2. A method for diagnosing an increased likelihood of developing a disease associated with G protein dysregulation comprising determining the presence of a genetic modification in a gene obtained from a subject which encodes the Gbeta3 subunit of the human G protein, wherein said genetic modification is a substitution of cytosine by thymine at position 1429 of SEQ ID NO:2.

3. The method of claim 2, wherein said genetic modification is at position 825 of SEQ ID NO:2.

4. The method of claim 2, wherein the disease associated with G protein dysregulation is for diabetes mellitus type 2, obesity and adiposity, hypercholesterolemia, coronary heart disease, myocardial infarction, sudden cardiac death, osteoporosis, atherosclerosis, neurodegenerative or cerebrovascular conditions, Alzheimer's disease which is based on the increased reactivity of the immune system and/or not developing an erectile dysfunction.

5. A method for diagnosing an increased likelihood of a woman developing a cardiovascular condition, comprising determining the presence of a genetic modification in a G protein beta3 subunit obtained from the woman, wherein said genetic modification is a cystosine to thymine substitution of position 1429 of SEQ ID NO:2.

6. The method of claim 5, wherein said genetic modification is at position 825 of SEQ ID NO:2.

7. A method for determining an increased risk of an individual for developing a disease associated with G protein dysregulation comprising comparing a gene sequence for the Gbeta3 subunit of the human G protein of the individual compares with a gene sequence of SEQ ID NO:2, wherein a correspondence between the sequences indicates an increased risk of disease being assigned to the individual.

8. The method of claim 7, wherein in the gene sequence of the individual corresponds with the gene sequence of SEQ ID NO:2 at position 825.

9. The method of claim 7, wherein to determine the risk of developing diabetes mellitus type 2, gene changes in the IRS1 gene (3931A variant; Gly971Arg), in the IRS2 gene, in the gene which codes for the p85 alpha regulatory subunit of PI3 kinase (1020 G -> A; codon 326 Met -> Ile), in the gene which codes for the beta3 adrenergic receptor (Trp64Arg), in the gene which codes for the beta2-adrenergic receptor (here especially Arg16Gly variant and the Gln27Glu variant), in the gene which codes for the tumor necrosis factor alpha and/or in the gene which codes for leptine or the leptine receptor, are further evaluated.

10. The method of claim 7, wherein to determine the risk of developing obesity and adiposity, gene changes in the IRS1 gene (3931A variant; Gly971Arg), in the gene which codes for the beta3 adrenergic receptor (Trp64Arg variant), and/or in the gene which codes for the beta2-adrenergic receptor (here especially Arg16Gly variant and the Gln27Glu variant) are further evaluated.

11. The method of claim 7, wherein to determine the risk of developing coronary heart disease and/or myocardial infarction, gene changes in the IRS1 gene (3931A variant; Gly971Arg) are further evaluated.

12. The method of claim 7, wherein to determine the risk of developing diseases which are associated with increased reactivity of the immune system, gene changes in the beta2-adrenergic receptor (here especially the Arg16Gly variant and the Gln27Glu variant) are further evaluated.

13. The method of claim 7, wherein to determine the risk of developing gestosis, gene changes in the gene coding for endothelial NO synthase (especially the Glu298Asp variant) are further evaluated.

14. The method of claim 7, wherein an increased risk of developing AIDS is assigned to homozygotic HIV-positive individuals.

15. The method of claim 14, wherein to determine the risk of developing AIDS, gene changes in the CCR5 gene are further evaluated and wherein a further

increased risk of developing AIDS is assigned to the homozygotic or heterozygotic individuals for the CCR5_32 polymorphism.

16. The method of claim 15, wherein to determine the risk of developing AIDS, gene changes in the CCR5 gene are further evaluated and wherein a further increased risk of developing AIDS is assigned to the individuals which carry the CCR5P1 allele.

17. The method of claim 14, wherein to determine the risk of developing AIDS, SDF1-3'UTR-801G-A polymorphism is evaluated and wherein a further increased risk of developing AIDS is assigned to the individuals which carry the SDF1-3'A allele.

18. A method for evaluating responsiveness of an individual to an in vivo pharmaceutical comprising evaluating the individual for a genetic modification in a gene encoding a Gbeta3 subunit of a protein, wherein the genetic modification is a substitution of cytosine by thymine at position 825 and/or at position 1429 of SEQ ID NO:2.

19. A method for evaluating responsiveness of an individual to in vivo to hormones, transmitters, neurotransmitters or pharmaceuticals which activate those G protein heterotrimers which contain the G protein subunits Gbeta3 and Gbeta3s and/or which stimulate the G protein subunit GalphaS comprising evaluating the individual for a genetic modification in a gene encoding a Gbeta3 subunit of a protein, wherein the genetic modification is a substitution of cytosine by thymine at position 825 and/or at position 1429 of SEQ ID NO:2..

20. The method of claim 18 or 19, further comprising determining the presence of the Arg16Gly variant and the Gln27Glu variant in the beta2 adrenergic receptor.

21. The method of claim 18, wherein pharmaceutical is erythropoietin.

22. The method of claim 18, wherein the pharmaceutical is an immunosuppressive and the development of hypertension during such therapy is evaluated.

23. The method of claim 22, wherein the immunosuppressive is cyclosporin.

24. The method of claims 19, wherein the pharmaceutical is for treatment and prevention of a migraine headache.

25. A method for evaluating responsiveness of an individual to treatment with beta-adrenoceptor blockers comprising evaluating the individual for a genetic modification in a gene encoding a Gbeta3 subunit of a human G protein, wherein the genetic modification is a substitution of cytosine by thymine position 825 and/or position 1429 of SEQ ID NO:2.

26. A method for evaluating responsiveness of an individual in treatment with a substance having prostoglandin E1 action comprising evaluating the individual for a genetic modification in a gene enclosing a Gbeta3 subunit of a human G protein, wherein the genetic modification is a substitution of cytosine by thymine position 825 and/or position 1429 of SEQ ID NO:2.

27. The method of claim 26, wherein the substance is prostaglandin E1.

28. An isolated and purified nucleic acid having a sequence complementary to the nucleic acid sequence as claimed in claim 1.

29. Use of a protein of the Gbeta3s subunit of the human G protein in recombined systems or after transfection in suitable cell lines for identifying chemicals except for antibodies, which inhibit the function of Gbeta3s.

30. A beta-3 subunit of a human G protein which has at most six WD repeat motives, wherein the Gbeta3s subunit has the amino acid sequence shown of SEQ ID NO:4.

31. A nucleic acid sequence coding for a protein as claimed in claim 30.

32. A nucleic acid sequence as claimed in claim 31, having the sequence of SEQ ID NO:4.

33. A process for producing a protein as claimed in claim 30, wherein a nucleic acid sequence as claimed in one of claims 31 or 32 is introduced into a host and expressed.

34. The process as claimed in claim 33, wherein expression takes place in immune cells of immune-deficient individuals.

35. The process as claim 34, wherein the individual is HIV-positive.

36. The process as claimed in claim 33, wherein expression is in human body cells.

37. Use of a nucleic acid sequence as claimed in claim 30 or 31 for producing a pharmaceutical for treatment of diseases which are associated with G protein dysregulation.

38. A transgenic animal comprising the nucleic acid sequence as claimed in claim 31 or 32 .

39. A nucleic acid sequence which is complementary to the nucleic acid sequence as claimed in claim 31 or 32.

40. An antibody directed against the protein of claim 30.